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Human Physiology at Extreme Altitudes on Mount Everest

John B. West

Summary. Extreme altitude presents an enormous physiological challenge to the human body because of severe oxygen deprivation. The American Medical Research Expedition to Everest was specifically designed to study man under these conditions, and successfully obtained physiological data above 8000 meters, including a few measurements on the summit itself. The results show that man can tolerate the extreme hypoxia only by an enormous increase in ventilation, which results in an alveolar partial pressure of carbon dioxide of 7.5 torr on the summit and an arterial *p*H of over 7.7. Even so, the arterial partial pressure of oxygen is apparently less than 30 torr, and maximum oxygen uptake is about 1 liter per minute. Additional measurements of ventilation, blood physiology, and metabolic and psychometric changes clarified how man responds to this hostile environment.

High altitude has always intrigued physiologists because of the remarkable ability of man and other animals to adapt to the hostile environment. When we ascend to elevations where the inspired partial pressure of oxygen (PO_2) falls to low levels, a whole series of compensatory changes take place in a process known as acclimatization. The changes include an increase in pulmonary ventilation, polycythemia, a rightward shift of the oxygen dissociation curve, an increase in the number of capillaries in peripheral tissues, and changes in oxidative enzymes within cells (*1*). As a result of these changes, lowlanders can spend extended periods of time at altitudes up to about 5300 m. This altitude also marks the highest habitation of permanent high-altitude dwellers, and such people, as in the South American Andes, show physiological changes that are in some ways similar to those of acclimatized lowlanders, although there are important differences (*1*).

Extreme altitudes, say above 6000 m, have evoked special interest in the past few years. A major milestone was the first ascent of Mount Everest (altitude 8848 m) without supplementary oxygen in 1978 by Messner and Habeler (*2*). Many physiologists thought that this feat could never be achieved, and the event was responsible for a surge of interest in the effects of extreme hypoxia on human physiology.

The American Medical Research Expedition to Everest in fall 1981 was designed to make the first measurements of human physiology above 8000 m. Data were successfully obtained above this altitude, and a few measurements were made on the summit itself. In addition, two laboratories were set up at 6300 and 5400 m, and a wealth of new information about man at extreme altitudes was obtained.

American Medical Research Expedition

There are two ways of studying the human response to prolonged exposure to low oxygen. One is to use a low-pressure chamber, but this has several disadvantages. For example, it is not clear whether subjects can tolerate confinement under reduced pressures for several weeks and remain physically fit. In addition, the psychological consequences of such confinement might complicate the results (*3*). Such an exposure is necessary to develop the acclimatization required in order to tolerate the low oxygen pressures that exist near the summit of Mount Everest.

A better solution is to use the natural laboratory of the mountain itself. However, it is difficult to accomplish scientific objectives on a regular mountaineering expedition, so the American Medical Research Expedition to Everest had an unusual design. The expedition had six highly experienced Himalayan climbers, including John P. Evans, climbing leader. Next, there was a group of six "climbing scientists," all of whom were strong climbers, but each was a doctor of medicine with an interest in high-altitude physiology. Their responsibility was to carry out the measurements at extreme altitudes. Finally, there was a third group of eight physiologists who worked

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in the two laboratories at Camp 2 (6300 m) and Base Camp (5400 m) (*4*).

Physiological measurements were carried out at four sites on the mountain (Fig. 1), these being largely determined by the topography. After a 3½-week trek from Kathmandu, Base Camp (5400 m) was set up at the head of Khumbu Glacier. A rigid prefabricated laboratory hut was erected here, and an extensive research program was carried out during the months of September and October 1981. Immediately behind Base Camp is the steep, unstable, treacherous Khumbu icefall. This leads to a relatively flat valley called the Western Cwm. The main laboratory (Camp 2) was located here at an altitude of 6300 m (Fig. 2). The laboratory was constructed of an aluminum frame covered by fiberglass blankets (*5*) with a plywood floor, and it was heated with propane and supplied with electrical power from solar panels and gasoline generators. A series of research projects were completed there during October. At the end of the Western Cwm is the headwall leading to the South Col of Everest and the final summit ridge. A few measurements were carried out at Camp 5, altitude 8050 m, just above the South Col. We had carried in a special laboratory tent to erect there, but high winds prevented this. Nevertheless, samples of venous blood were taken and some sleep electrocardiograms were measured.

Most ambitious of all were the experiments planned for the summit. We recognized that many expeditions to Mount Everest are unsuccessful in reaching the summit and that to plan for physiological measurements there was tempting fate. However, Pizzo successfully obtained the first direct measurement of barometric pressure, and collected several samples of alveolar gas (air from the depths of the lung). These were obtained with a specially designed sampler. The climber exhaled into a mouthpiece and pulled a lever that opened the valve of a small, evacuated aluminum can. This trapped the end-expired gas, which was brought back to San Diego for analysis by mass spectrometer. In addition, continuous electrocardiograms were obtained on two climbers who reached the summit, and maximum exercise ventilation was also measured.

Physiology on the Summit of Everest

Barometric pressure. Great trouble was taken to obtain accurate measurements of barometric pressure because previous predictions had indicated that maximum oxygen uptake would be extremely sensitive to changes in inspired PO_2 (*6*). At Base Camp, measurements were made with a Fortin mercury barometer specially shortened for the expedition. Above Base Camp, most of the measurements were made by using a small portable barometer with a crystal-sensor transducer of high intrinsic accuracy (*7*). Barometric pressure at 5400 m was 400.4 ± 2.7 torr (mean ± standard deviation), and this fell to 283.6 ± 1.5 torr at 8050 m. The one measurement obtained on the summit was 253.0 torr. The altitudes of these three sites are accurately known from surveys.

All these pressures are substantially above those predicted from the International Civil Aviation Organization Standard Atmosphere (*8*) for these altitudes. For example, our reading at the summit was 17 torr higher than that given by the Standard Atmosphere, which has been extensively used by physiologists to predict the deleterious effects of hypoxia at great altitudes. The reason for the higher pressures on Mount Everest is that barometric pressures 4 to 16 km above sea level are markedly latitude-dependent (*9*) because of the presence of a large mass of cold air in the stratosphere above the equator that results from convective and radiation phenomena. The summit of Everest is at 28°N, and it therefore enjoys this higher pressure. The Standard Atmosphere is a model that was never

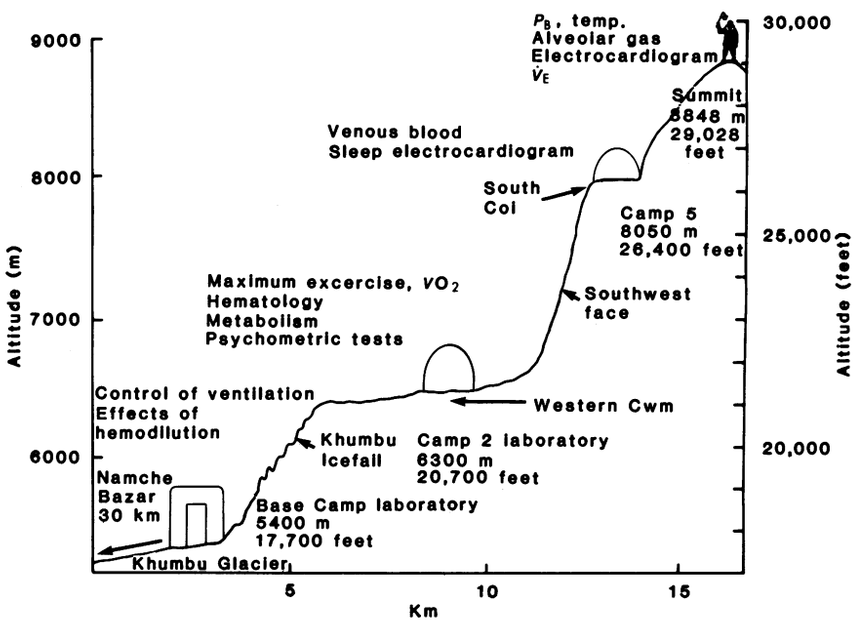

Fig. 1. Research projects carried out during the expedition. The four sites for experiments were determined by the topography of the south aspect of Mount Everest. P_B, barometric pressure; $\dot{V}_E$, exercise ventilation.

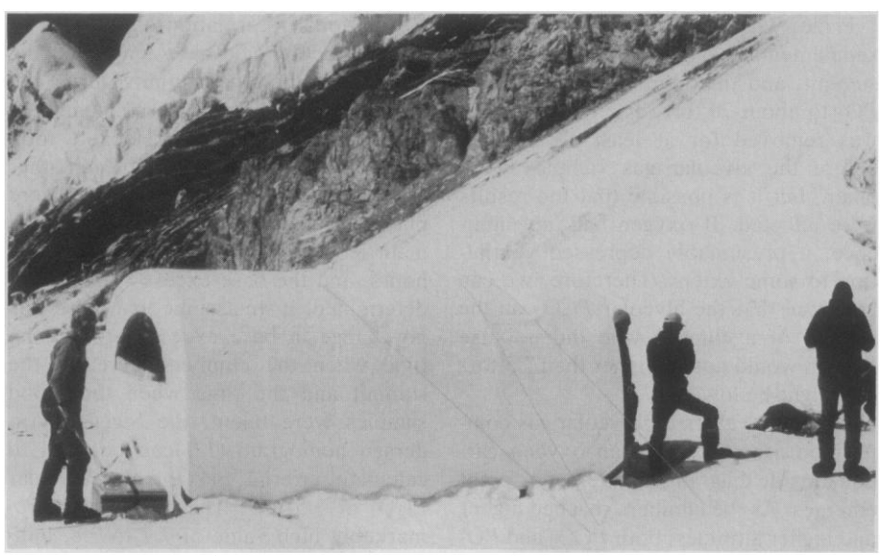

Fig. 2. Main laboratory at Camp 2, altitude 6300 m.

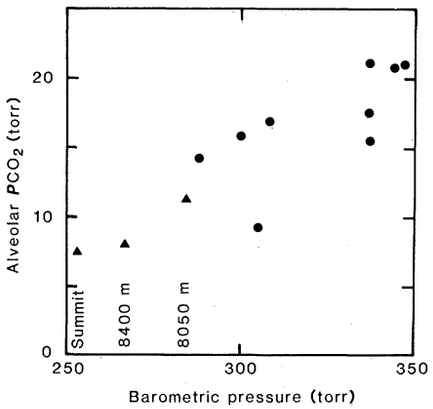

Fig. 3. Relation between alveolar PCO_2 and barometric pressure at extreme altitudes. Note the remarkably low value at the summit (about 7.5 torr). [From (*10*)]

meant to be used to predict pressure at any particular location. It is interesting that if the barometric pressure at the summit were not increased by this equatorial bulge, it would not be possible to reach the summit without supplementary oxygen (*6*).

Pulmonary gas exchange. Thirty-four valid alveolar gas samples were collected above 8000 m (*10*). The mean PCO_2 at Camp 5 was 11.0 ± 2.0 torr (N = 27), and this fell to a mean value of 8.0 (N = 3) at 8400 m. The mean of four samples on the summit was 7.5 torr. The normal sea level value is 40 torr. Figure 3 shows the mean PCO_2 at the three altitudes where we obtained data (triangles) and other measurements made by previous expeditions at barometric pressures below 350 torr (circles). There is an approximately linear decrease in alveolar PCO_2 as barometric pressure falls, with astonishingly low values at extreme altitudes, indicating the extreme hyperventilation characteristic of successful climbers.

Pizzo, who took these samples, used supplementary oxygen en route to the summit, and this increased his inspired PO_2 to about 70 torr. The oxygen mask was removed for at least 10 minutes before the alveolar gas samples were taken, but it is possible that the results were affected. If oxygen had any influence, it presumably depressed ventilation to some extent. Therefore, we can conclude that the alveolar PCO_2 on the summit of a climber who did not use oxygen would not be higher than 7.5 torr but might be lower.

When the values for alveolar gas composition are plotted on an oxygen–carbon dioxide diagram, an interesting point emerges. As the climbers reached higher and higher altitudes, both PCO_2 and PO_2 fell, the former by the hyperventilation and the latter by the reduced inspired PO_2. However, when PO_2 had fallen to about 35 torr at about 6500 m, there was essentially no further reduction in PO_2 as altitude increased. In other words, the increasing hyperventilation was sufficient to defend alveolar PO_2 at about 35 torr. This appears to be one of the most important ways in which the body protects itself against the severe hypoxia of extreme altitudes. Incidentally, it is likely that not all subjects can respond to increasing altitudes with this degree of hyperventilation. But then, not everyone can reach the summit of Mount Everest, and these two facts are probably connected.

What can be said about arterial PO_2 under these conditions? With present technology it is impossible to take arterial blood on the summit. However, useful information can be obtained by calculating the change in PO_2 along the pulmonary capillary, the so-called Bohr integration (*11*). Such a calculation on a subject at sea level shows a very rapid rise in the PO_2 of pulmonary capillary blood early in the capillary and essentially no PO_2 difference between alveolar gas and end-capillary blood. However, when the same calculation is made for a climber on the summit by using the measured alveolar gas values and blood samples taken at 8050 m, the results are strikingly different. The PO_2 in the pulmonary capillary rises slowly, reaching only about 28 torr at the end of the capillary. There is a large PO_2 difference of about 7 torr between alveolar gas and end-capillary blood, indicating diffusion limitation of oxygen transfer. Such limitation never occurs in the normal lung at sea level and is a striking indication of how poorly adapted the human lung is for these extreme environmental conditions.

Additional information allowed us to determine the acid-base status of the arterial blood on the summit. During the morning after their successful climb to the summit, Pizzo and Hackett took samples of venous blood from each other at Camp 5 (8050 m). These samples were placed on ice and carried down to the main laboratory (6300 m) within a few hours, and the base excess values were determined. If we assume that there was no change in base excess between the time when the climbers were on the summit and the time when the blood samples were taken, the Siggaard-Andersen nomogram (*12*) can be used to calculate arterial pH for the alveolar PCO_2 of 7.5 torr. The result is the remarkably high value of 7.7 to 7.8, indicating extreme respiratory alkalosis resulting from hyperventilation. The reduction in base excess at these extreme altitudes was much less than we had predicted, and it appears that there was virtually no further decrease in base excess above 6300 m. More work needs to be done on this apparent failure of the kidney to excrete additional bicarbonate under these conditions.

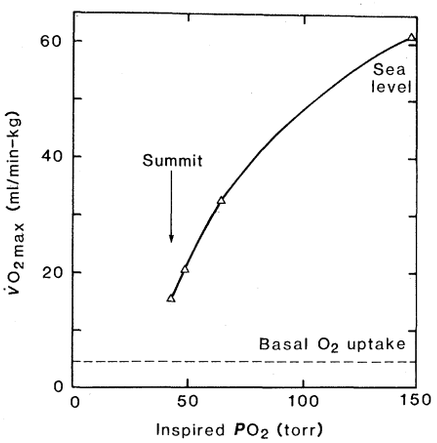

Fig. 4. Relation between $\dot{V}O_{2\,max}$ and inspired PO_2 as altitude increased. Note that at a PO_2 corresponding to the summit, $\dot{V}O_{2\,max}$ was just over 1 liter/min for a 70-kg man. The two lowest values of inspired PO_2 were obtained by giving acclimatized subjects low oxygen concentrations to breathe at 6300 m.

Table 1 summarizes the alveolar gas and arterial blood values for a resting climber on the summit as best as can be determined from available data.

Maximum exercise. An interesting question concerns the maximum oxygen consumption of the body under these conditions of extreme oxygen deprivation. Extrapolation from previous measurements at somewhat lower altitudes suggests that the maximum oxygen consumption on the summit might be near the basal oxygen requirements (*6*). Also, the fact that climbers without supplementary oxygen got to within 300 m of the summit as early as 1924 but did not reach it until 1978 suggests that man is very near his limit of oxygen tolerance.

It was not possible to make direct measurements of maximum oxygen uptake on the summit. However, essentially equivalent data were obtained by having two well-acclimatized subjects exercise maximally on a bicycle ergometer in the Camp 2 laboratory (6300 m) while breathing 14 percent oxygen. Under these conditions the inspired PO_2 was 43 torr, the same as on the summit. Measurements were also made with subjects breathing 16 percent oxygen and ambient air, and the results were compared with sea level measurements made using the same techniques.

Figure 4 shows maximum oxygen uptake ($\dot{V}O_{2\ max}$) plotted against inspired PO_2 as the altitude increased. At an inspired PO_2 of 64 torr (altitude 6300 m, ambient air), $\dot{V}O_{2\ max}$ was reduced to about 50 percent of the value at sea level. At an inspired PO_2 corresponding to the summit, $\dot{V}O_{2\ max}$ was reduced to about 23 percent of the sea level value, or about 1.1 liter/min. This $\dot{V}O_{2\ max}$ on the summit is clearly very limiting, being equivalent to that achieved by someone walking slowly on level ground at sea level. However, it is apparently sufficient to explain how Messner and Habeler reached the summit without supplementary oxygen. They reported their climbing rate as only 2 m/min near the summit and, if we take the weight of a climber plus equipment to be 100 kg, the average work rate is then 200 kg/min. This is therefore appreciably less than the 300 kg/min corresponding to the $\dot{V}O_{2\ max}$ of 1.1 liter/min. In fact, both our subjects were able to sustain a work rate of 450 kg/min for 3 minutes, although there was no increase in oxygen uptake, indicating that they were merely incurring a larger oxygen debt.

Other Measurements at Extreme Altitudes

Space does not permit a full description of the extensive research program of the expedition, so only a summary of some of the other results is given here.

Control of ventilation. Studies of the control of ventilation in lowlanders and Sherpas in both the awake and sleeping states were carried out by Lahiri. A striking observation was that although all lowlanders showed periodic breathing (Cheyne-Stokes respiration) during sleep at 5400 and 6300 m, almost all the Sherpas did not. The only exception was one Sherpa who lived at low altitude. The pattern of periodic breathing in the sojourners during sleep was altered when the inspired PO_2 was transiently raised. Although some fluctuations in amplitude of the tidal volume usually persisted, any apneic periods disappeared.

A possible explanation for the lack of periodic breathing in the Sherpas is that it is related to their blunted hypoxic ventilatory response, which reduces the gain of the feedback loop from the peripheral chemoreceptors and thus prevents the instability that otherwise occurs. In keeping with this blunted hypoxic ventilatory response, Sherpas like Andean high-altitude natives have a slightly higher arterial PCO_2, both at rest and during exercise, slightly lower arterial PO_2 (although this reduction may be mitigated by a higher respiratory exchange ratio), and lower exercise ventilation than sojourners at high altitude. Sojourners at high altitude hypoventilate when given 100 percent oxygen, but Sherpas increase their ventilation. The reason for this is unknown.

Table 1. Alveolar gas and arterial blood values on the summit of Mount Everest.

Altitude	Barometric pressure (torr)	Inspired PO_2 (torr)	Alveolar PO_2 (torr)	Arterial		
				PO_2 (torr)	PCO_2 (torr)	*p*H
8848 m (summit)	253	43	35	28	7.5	> 7.7
Sea level	760	149	100	95	40	7.40

Comparisons of ventilatory responses to hypoxia at sea level and high altitude in expedition members were carried out by Schoene and Lahiri. They found that the magnitude of the hypoxic ventilatory response at sea level was generally predictive of the response during rest at 5400 m and was correlated with exercise ventilation at 6300 m. It also appeared that performance of the expedition members on the mountain, as judged by maximum altitude attained or highest camp slept at, was correlated with the magnitude of the hypoxic ventilatory response measured at sea level. There have been previous intimations of this relation, and it is of considerable interest because it gives a potential predictor of performance at extreme altitude that can be obtained at sea level.

Blood physiology. These measurements were carried out by Winslow and Samaja. Polycythemia was seen, the mean hemoglobin concentration at 6300 m being 18.8 g/dl with a mean hematocrit of 53.4 percent. There was also a small increase in mean corpuscular hemoglobin concentration, which may reflect some dehydration (difficult to avoid at these altitudes). Although the polycythemia was obvious, it was not apparently as extreme as seen in some previous expeditions (*13*). The concentration of 2,3-diphosphoglycerate showed a mean increase of about 0.2 mole per mole of hemoglobin, and this increase caused a rightward shift of the oxygen dissociation curve, the P_{50} (partial pressure of oxygen at which hemoglobin is half-saturated) increasing by 1 to 2 torr. However, arterial *p*H exceeded 7.4 at 6300 m because of a partially uncompensated respiratory alkalosis, and the increased *p*H caused a leftward shift of the oxygen dissociation curve so that P_{50} in vivo was actually lower than the control value at sea level. This in vivo P_{50} became progressively lower as altitude increased because of more severe alkalosis, and the calculated value for one subject on the summit was less than 20 torr. Measurements on man and animals and theoretical studies suggest that this leftward shift of the oxygen dissociation curve is advantageous at high altitude because it enhances the loading of oxygen by the pulmonary capillary (*14*).

The effects of reducing the hematocrit to lower levels by hemodilution was studied by Sarnquist, Schoene, and Hackett. This experiment was carried out because it has been suggested that the polycythemia of high altitude may be deleterious due to the associated increase in blood viscosity. In four subjects with hematocrits of 58 percent or more, values were reduced to 50 percent by bleeding while blood volume was kept constant by an infusion of albumin solution. No changes in maximum exercise capacity or psychometric performance could be demonstrated, suggesting that this degree of polycythemia is not a useful adaptation. Winslow reported similar findings in a study on permanent high-altitude residents in the Peruvian Andes.

Metabolism. Extensive metabolic measurements were carried out by Blume and Boyer. At 6300 m there was a striking loss of body weight that included both body fat and muscle mass, as evidenced by reductions in limb girth. Factors responsible for the loss of weight included loss of appetite and significant reductions in both fat and xylose absorption. It is possible that alterations in protein metabolism also occurred.

Blood samples were frozen and brought back to the United States for analysis. Serum glucose in the fasting state was reduced at 6300 m, and the glucose tolerance curve was much flatter than at sea level. Serum insulin concentrations after glucose loading were also lower than at sea level. Glucagon concentrations both in the fasting state and after glucose loading were normal. It appears that intestinal absorption of glucose may have been impaired although acceleration of glucose removal from the blood was a possible reason. Lipid studies showed increased fasting serum triglycerides, which, together with the de-

crease in skin fold fat, suggests that fat was being mobilized to serve as an energy source. Fasting serum protein levels were also increased, consistent with increased protein metabolism.

Thyroid hormones showed several significant changes (*15*). Serum thyroxine and free thyroxine index increased significantly and progressively with altitude. Triiodothyronine concentrations also increased, as did the fasting level of thyroid-stimulating hormone (TSH) in spite of the marked elevation in thyroxine. Intravenous injection of thyroid releasing hormone in fasting subjects at 6300 m produced abnormally high TSH responses, suggesting that the pituitary feedback set point for TSH secretion was altered.

In studies of catecholamines, elevated concentrations of norepinephrine were found while epinephrine remained normal. In spite of this, serum cortisol levels were shown to be normal at 6300 m. An interesting finding was that growth hormone concentrations remained at sea level values in all but two members of the expedition. However, in these two members, who both lost 15 kg, the level of growth hormone in serum increased fivefold.

The renin-angiotensin system was studied by Milledge because of previous evidence that alterations in this system may contribute to the fluid retention sometimes seen at high altitude. It was found that, although plasma renin activity was increased severalfold after exercise at 6300 m, plasma aldosterone concentrations were only slightly increased. Since serum angiotensin converting enzyme activity was normal, it is possible that there was a reduction in the density of angiotensin II receptors on the adrenal, or induction of enzymes that degrade angiotension II.

Cerebral function. In a study designed by B. D. Townes and T. H. Hornbein at the University of Washington, a series of psychological tests were carried out before, during, and after the expedition. These included the Halstead Reitan neuropsychological test battery (*16*) and tests of memory and coordination. Measurements were made at altitudes of 5400, 6300, and 8050 m, although only the simplest tests were done at extreme altitudes.

The most significant abnormality was a reduction in finger-tapping speed, a test in which the subject depresses a lever with one finger as rapidly as possible over a period of 10 seconds for three or more intervals. Of 16 expedition members, 15 showed continued impairment after the expedition, and the abnormality was still present in 13 subjects 1 year later. Motor impairment after prolonged periods at high altitude has also been found by other investigators (*17*). The reason is unclear but may be related to cerebellar dysfunction caused by prolonged, severe hypoxia.

A significant decline was also found in verbal learning and short-term memory as measured by the Wechsler memory scale (*18*). However, when the test was repeated 1 year later, performance had returned to the preexpedition level. There was also a significant impairment in expressive language as measured by the aphasia screening test but again performance was normal 1 year later.

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4. Members of the expedition were: *climbers* J. P. Evans (deputy leader—climbing), D. P. Jones, C. Kopczynski, J. Lowe, G. E. Porzak, and M. H. Weis; *climbing scientists* S. Boyer, D. J. Graber, P. H. Hackett, C. J. Pizzo and F. H. Sarnquist, R. B. Schoene; and *scientists* F. D. Blume (deputy leader—logistics and finance), S. Lahiri, K. H. Maret, J. S. Milledge, R. M. Peters, Jr., M. Samaja, J. B. West (expedition leader), and R. M. Winslow. R. A. Korich was Base Camp manager. The expedition was supported by 42 high-altitude Sherpas and two liaison officers. Three Americans (Kopczynski, Pizzo, and Hackett) and two Sherpas (Sundare and Yong Tensing) reached the summit.
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19. This ambitious scientific program could not have been successfully carried out without the full cooperation of all members of the expedition. Supported by PHS grant RO1-HL 24335 and contract NO1-HR-2915 and grants from the American Alpine Club, American Lung Association, National Geographic Society, National Science Foundation, Servier Laboratories (Paris), The Explorers Club, and the U.S. Army Research and Development Command.